AMENDMENTS TO THE SPECIFICATION:

Pursuant to 37 C.F.R. § 1.121, please amend the specification as follows:

Please replace the paragraph beginning at page 217, line 32 with the following amended paragraph:

Tyr-31 in the mature sequence of CTLA-4BP 5x4-12c (as measured from the N-terminus of the mature domain, using the amino acid position and numbering corresponding to the amino acid sequence of hB7-1 (SEQ ID NO:278) was replaced by histidine (i.e., Tyr31His ~~Try31His~~); the Tyr31His substitution was also present in a number of selected recombinant clones with preferential binding to CTLA-4, whereas all selected clones with preferential binding to CD28 retained the tyrosine at that position. Tyr-31 was also present at an equivalent position in all of the mature parental sequences. Tyr-31 of the mature CTLA-4BP and mature hB7-1 sequences (as measured from the N-terminus of the mature domain, using the amino acid position and numbering corresponding to the amino acid sequence of hB7-1) corresponds to Tyr-65 of the full-length CTLA4-BP and hB7-1 sequences, respectively (as measured from the N-terminus, which includes the signal peptide sequence). DNA sequence analysis of CTLA-4BP 5x4-12c showed a mutation in the codon corresponding to amino acid position 31 as measured from the N-terminus in mature hB7-1 – TAC – to codon CAC (at the corresponding position in mature form of CTLA-4BP 5x4-12c). Interestingly, it has been suggested that Tyr31 in human B7-2 may be replaced by phenylalanine without any apparent change in the ligand binding affinities (see, e.g., Freeman, G.J. et al. (1993) Science 262: 909-11); Azuma, M. et al. (1993) Nature 366:76-9), whereas a Tyr31Ala substitution in hB7-1 appears to completely abolish the binding of hB7-1 to both CD28 and CTLA-4 (see Peach, R.J. et al. (1995) J Biol Chem 270:21181-7). The present data demonstrate that the Tyr31His substitution, at least when present in the context of the shuffled CTLA-4BP sequence, does not significantly affect interaction with CTLA-4, whereas this mutation appears to contribute to the loss in binding to CD28, further supporting the suggestion that this residue plays an important role in the ligand binding of B7-1. Information regarding the three-dimensional structures of CD28BP and CTLA-4BP is useful in characterizing further the amino acid residues and structures that contribute to the preferential binding of NCSMs to their two respective ligands.